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Heritability of Obsessive-Compulsive Symptom Dimensions

Daniël S. van Grootheest,[1,2]* Dorret I. Boomsma,[1] John M. Hettema,[3] and Kenneth S. Kendler[3]

[1]*Department of Biological Psychology, Vrije Universiteit Amsterdam, Amsterdam, The Netherlands*
[2]*Department of Psychiatry, VU University Medical Center, Amsterdam, The Netherlands*
[3]*Department of Psychiatry, Virginia Institute for Psychiatric and Behavioral Genetics, Virginia Commonwealth University, Richmond, Virginia*

Recent research has shown that obsessive-compulsive symptoms (OCS) differ remarkably among patients and can be divided into several symptom dimensions. OCS are influenced by genetic components, but it is unknown to what extent these symptom dimensions are heritable. The phenotypic heterogeneity also raises the question to what extent the symptom dimensions are influenced by specific or shared genetic factors. We studied a population sample of 1,383 female twins from the Virginia Twin Registry. OCS was measured by a questionnaire with 20 items from the Padua Inventory. After factor analysis, three reliable OC symptom dimensions were retained: Rumination, Contamination, and Checking. These OC dimensions were analyzed with multivariate genetic models to investigate both the overlap and uniqueness of genetic and environmental contributions underlying OC symptom dimensions. The multivariate common pathway model provided the best description of the data. All symptom dimensions share variation with a latent common factor, that is, OC behavior. Variation in this common factor was explained by both genes (36%) and environmental factors (64%). Only the Contamination dimension was influenced by specific genes and seemed to be a relatively independent dimension. The results suggest that a broad OC behavioral phenotype exists, influenced by both genes and nonshared environment. In addition, we found evidence for specific genetic and environmental factors underlying the Contamination dimension. Use of the Contamination dimension could therefore provide a powerful approach for the detection of genetic susceptibility loci that contribute to OCS.

KEY WORDS: obsessive-compulsive disorder; OCD; Padua Inventory; twin study; structural equation modeling

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INTRODUCTION

In recent years, research on obsessive-compulsive disorder (OCD) showed that obsessive-compulsive (OC) symptoms are remarkably heterogeneous, so that two patients with this diagnosis can display completely non-overlapping symptom patterns [Mataix-Cols et al., 2005]. This is in contrast to the current concept adopted by the DSM-IV, which defines OCD as a unitary nosological entity [American Psychiatric Association, 1994]. This variability in phenotype may impact not only the findings of clinical, natural history and treatment response studies, but also complicate genetic studies and the search for vulnerability genes [Miguel et al., 2005]. One suggested approach to reconceptualize OCD or Obsessive-Compulsive Symptoms (OCS) is the use of OC symptom dimensions [Miguel et al., 2005]. OCD or OCS appears to encompass at least four consistent and temporally stable symptom dimensions [Mataix-Cols et al., 2005]. By considering these OC symptom dimensions as quantitative components of a more complex OC phenotype, a dimensional approach could provide a more powerful approach for the detection of genes or environmental risk factors that contribute to OC behavior [Miguel et al., 2005]. However, before using symptom dimensions in linkage or association analyses, it is important to examine the extent to which these symptom dimensions are heritable.

Alsobrook et al. [1999] were the first to use OC symptom dimensions in a family study. They reported that the relatives of OCD probands who had high scores on either the Yale-Brown Obsessive-Compulsive Scale (Y-BOCS) factors "aggressive/sexual/religious obsessions and related compulsions" or "symmetry/ordering" were twice as likely to have first-degree family members with OCD compared to individuals with low scores on these factors. Leckman et al. [2003] examined the familiality of OC symptom dimensions in sib pairs affected with Tourette syndrome. Significant correlations were observed between sib pairs as well as mother-child pairs for the "aggressive/sexual/religious obsessions and checking compulsions" factor, and for the "symmetry/ordering obsessions and compulsions" factor. Recently, Hasler et al. [2007] evaluated the familiality of different Y-BOCS dimensions within 418 sib pairs. Robust sib-sib intraclass correlations of around 0.2 were found for two of the four Y-BOCS factors: "hoarding obsessions and compulsions", and "aggressive/sexual/religious obsessions and checking compulsions". Smaller, but still significant, familiality was found for "contamination/cleaning," and "symmetry/ordering/arranging."

To disentangle genetic and environmental factors, twin or adoption studies are needed. No adoption studies examining OCD have been published. Twin studies of OCD have evolved from case-studies with patients with OCD to large samples of unselected subjects using the whole distribution of OC symptoms [Van Grootheest et al., 2005]. This last approach was first used by Clifford et al. [1984] who examined 419 twin

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*Correspondence to: Daniël S. van Grootheest, Vrije Universiteit, Department of Biological Psychology, Van der Boechorststraat 1, 1081 BT Amsterdam, The Netherlands.
E-mail: Ds.van.grootheest@psy.vu.nl

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pairs of monozygotic (MZ) and dizygotic (DZ) twins with the Leyton Obsessional Scale. The heritability of OCS was estimated to be 47%. The only other large study using unselected adult twins was published by Jonnal et al. [2000]. They examined 527 female twin pairs and carried out a factor analysis on 20 Padua Inventory items. Two major factors were used in the genetic analysis, one factor which described thoughts and one which described actions, for example, obsessions and compulsions. Heritabilities of 33% and 26% for obsessions and compulsions, respectively, were found. Recently, Van Grootheest et al. [2007] obtained the Young Adult Self Report Obsessive-Compulsive Subscale from a group of 5893 mono- and dizygotic twins, and 1,304 additional siblings and found a moderate heritability of 39% for men and 50% for women.

A next step would be to use OC symptom dimensions in an epidemiological twin sample, allowing one to investigate the genetic and environmental factors underlying different OC symptom dimensions. The (most) heritable symptom dimensions may be useful as a refined phenotype for further linkage or association studies. In this study we present multivariate analyses of the OCS data described by Jonnal et al. [2000]. Instead of heritabilities of the classic obsession/compulsion factor model as originally reported, we present results of multivariate genetic analyses of empirically defined symptom dimensions, giving the opportunity to investigate both the overlap and uniqueness of genetic and environmental contributions underlying OC symptom dimensions. We aim to address three major questions:

(1) Can distinct dimensions within OCS be found in a general population sample of women?
(2) What role do genetic and environmental factors play in the etiology of these OC symptom dimensions?
(3) Are different symptom dimensions influenced by the same or by different genetic factors?

MATERIALS AND METHODS

Sample

Sample characteristics are extensively described in the publication of Jonnal et al. [2000]. Briefly, participants in this study were from a population sample of Caucasian female twins from the Virginia Twin Registry [Kendler and Prescott, 2006]. Self-report questionnaires on OC items were mailed to 1942 twins of whom 1382 returned completed questionnaires, the subjects of the current analyses. Zygosity was determined by analysis of a questionnaire and, when necessary, by DNA polymorphisms. Zygosity classifications were more than 95% accurate. The group of 1382 twins consisted of 524 complete pairs (331 MZ and 193 DZ pairs), and 334 twins whose co-twin was not assessed (175 MZ and 159 DZ twins). Their mean age was 36.6 (SD 8.4).

Scale

Twenty items of the Padua Inventory (PI) [Sanavio, 1988] were included in a self-report questionnaire. Items were chosen from all four OC dimensions of the original 60-item PI scale based on their factor loadings but also to maintain a diversity of item content. Participants were asked to respond positively or negatively to each item (yes or no) whether or not it described them. The PI is a comprehensive self-report measure for assessing symptoms of OCS. It was developed by Sanavio [1988] to obtain the most important and frequent types of obsessional complaints. From this original 60-item PI, a 41-item, the Padua Inventory Revised (PI-R) [Van Oppen et al., 1995] and a 39-item version, the Padua Inventory-Washington State University Revision (PI-WSUR), have been developed by examining the factorial structure of the PI and deleting items that were poor or impure measures of these factors. The PI-R was the first study on PI items that used also data of OCD patients, instead of data of a non-clinical sample only, like Sanavio et al. (1988) (original 60-item version) and Burns et al. (39-item revision version). Both revised versions contain almost similar items, but only the PI-R is still frequently used in research. The 41 items of the PI-R form five subscales or symptom dimensions, represent symptom categories that are commonly found in OCS: Impulses (or Aggressive/Harm Obsessions), Washing (or Contamination), Checking, Rumination, and Precision [Van Oppen et al., 1995; Denys et al., 2004]. The 20 items used in this study did not contain any Precision items. Van Oppen et al. [1995] reported good to excellent internal consistency for the full scale (range = 0.89–0.92), and the subscales (range = 0.66–0.89) in a group of patients with OCD, patients with other anxiety disorders, and a general population sample. OCD patients scored remarkably higher on the full PI-R and the subscales, than patients with other anxiety disorders and general population controls [Van Oppen et al., 1995]. Van Oppen et al. [1995] also found that the factorial structure of the PI-R is invariant across the OCD patient group, the anxiety patient group and the general population group. In other words, they found the same factorial structure in OCD patients and general population controls.

Statistical Analyses

Exploratory factor analysis was performed on the 20 items of the PI to investigate different dimensions of OC. Mplus [Muthén and Muthén, 2005] was used to perform the factor analysis with the categorical data analysis option. With this option, tetrachorical correlations are generated as basis for the factor structure. To correct for dependency of the data, we only used data from one twin, randomly chosen, per family in the factor analysis. We used an oblique rotation for the factor analysis, which allows components to correlate. We examined the scree plot and only factors with an eigenvalue of higher than 1 were retained. In accordance to Stevens [1996], factor loadings higher than 0.16 were regarded as significant for our sample size. Furthermore, only factors with a minimum of three items were interpreted. Instead of using factor scores, we summed the PI items with the highest loadings for the different factors. Using sum scores have the advantage that they can be easily reproduced by others and do not depend for their weights on one particular data set.

For example, when four questions scored the highest on factor one, we summed up the answers of those four questions for every twin. The same holds for the questions which scored highest on factor two, etc. These summed scores per dimension were used for subsequent genetic analyses. Internal consistency of each dimension was evaluated using Cronbach's alpha.

Genetic and environmental influence on the OC symptom dimensions were estimated using structural equation modeling. The influence of the relative contributions of genetic and environmental factors on individual differences in OC symptoms can be inferred from the different levels of genetic relatedness of MZ and DZ twins. Variance in OC symptom scores may be due to additive genetic effects (A), shared environmental effects (C), and nonshared environmental (E) effects. Because MZ twins share all their segregating genes, the genetic effects are perfectly correlated in MZ twins. DZ twins correlate 0.5, because DZ twins share on average half of their segregating genes. Shared environmental effects, environmental experiences that make twins from a pair similar in their liability to OC symptoms, correlate 1.0 within both MZ and DZ twins. Nonshared environmental effects are, by

definition, uncorrelated in both MZ and DZ twin pairs and include both the effect of individual experiences and measurement error. Because the distributions of the OC symptom dimensions were non-normally divided, characterized by skewness, that is, many respondents scored zero, we used categorical data analysis within Mx [Neale et al., 2003]. In this approach, a liability threshold model is applied to the ordinal scores, using a threshold to define affection status. It is assumed that a person is "unaffected" if his or her liability is below this threshold or "affected" if above this threshold. In the present study, a cutoff score of one was used to gain roughly two groups of the same size. This means that a person is "unaffected" for a symptom dimension if they possessed a score of zero on this symptom dimension or "affected" with a score of one or higher.

After fitting a fully saturated model, a model with all correlations and thresholds estimated freely, we fitted both independent and common pathway multivariate models to investigate the pattern of covariation among the OC dimensions and their relation to the construct OC behavior. The independent pathway model specifies common factors of A, C and E loadings on all the outcome measures (e.g., OC dimensions). Besides these common factors, it allows separate A, C and E decompositions of each observed OC dimension. To investigate whether the OC dimensions define a single construct of OC behavior, a common pathway model was also fit. In this model, both genes and environment are assumed to contribute to one latent (unmeasured) variable (e.g., OC behavior) which is responsible for the observed covariation between the scales. Genetic and environmental factors specific to each OC dimension are also incorporated in the model. When fitting models to ordinal data using a threshold approach, a constraint on the total latent variance is needed. For the independent pathway model, we constrained the total variance for each of the three dimensions to equal 1. For the common pathway model also the variance of the latent common phenotype was constrained to be 1. For more detailed information about independent and common pathway models see Martin and Eaves [1977] and Kendler et al. [1987].

We tested whether a model could be simplified by dropping one or more latent factors. The non-shared environmental factor was never dropped from the model, because, in addition to non-shared environmental experiences, this factor includes measurement error. The models were fitted to raw data with Mx [Neale et al., 2003] by the method of maximum likelihood estimation. This allowed the use of all twin data, including those without an interviewed co-twin. Goodness-of-fit was assessed by likelihood-ratio chi-square (χ^2) tests. These tests compare the differences between two times the log likelihood of a full model and a restricted nested model. This difference is distributed as χ^2, and the degrees of freedom (df) for this test are equal to the difference between the number of estimated parameters in the full model and that in the restricted model. More technical details of genetic model-fitting analyses are reviewed elsewhere [Neale and Cardon, 1992].

RESULTS

Factor Analysis

The results of the factor analysis using the 20 PI items were unclear and showed a difficult to interpret five-factor solution. We then decided to include only those Padua Inventory items which were used in the 41-item PI-R [Van Oppen et al., 1995]. Of the 20 items we collected, 17 met this criterion. Interestingly, the factor analysis of these 17 items showed an interpretable four-factor solution, which explained 46.6% of the variance (Table I). Inspection of the items included in these factors suggested that the components represented (1) Rumination, (2) Contamination, (3) Impulses or Aggressive/Harm Obsessions, and (4) Checking. The internal consistency of each factor was 0.67, 0.62, 0.48, and, 0.64 respectively. The factor Impulses clearly showed a lower internal consistency. Further inspection of this factor revealed that more than 90% of the participants scored 0 on this symptom dimension, which caused very low variation within this factor. We decided not to include this factor in our genetic analyses. Table II shows the phenotypic correlations between the dimensions Rumination, Contamination, and Checking.

Genetic Analyses

Tetrachoric twin correlations, both within dimensions and across dimensions, are seen for both zygosity groups in Table III. For all dimensions, MZ correlations were higher than DZ correlations, indicating the influence of genetic factors on OC dimensions (diagonal). However, for the factor Checking, shared environmental factors also seem important, because the MZ correlation is less than twice the DZ correlation. The cross-dimension twin correlations (off-diagonal), that is, the correlation between a OC dimension of the first-born twin with a different OC dimension of the second-born twin and vice-versa, give insight into the role of genes and environment in explaining the sources of the correlation between dimensions. Here we also see that MZ cross-dimension correlations are larger than DZ cross-dimension correlations. This suggests that genetics may help explain the overlap between the different dimensions. However, for the dimensions Rumination and Checking, shared environmental factors also are important, given that the MZ correlation is only slightly larger than the DZ correlation.

In the saturated model, we were able to constrain the thresholds for all three factors to be equal in both twins from a pair, and in both MZ and DZ pairs ($\chi^2(9) = 13.2$, $P = 0.16$). Compared with the multivariate fully saturated model (Table IV), the independent pathway fitted well to the data ($\chi^2(24) = 34.4$, $P = 0.08$). The common pathway model structure is different from that of the independent pathway model (it introduces a latent variable) and can be formally tested as a nested sub-model. Comparing the fit of the common pathway model to the independent pathway model produced a non-significant chi-square test ($\chi^2(4) = 6.0, P = 0.20$). This indicates that the more restrictive common pathway model provides a more parsimonious explanation than does the independent pathway model. The common pathway model is therefore the model of choice. Figure 1 displays the common pathway model with the estimates of the structural parameters. The total variance of the latent phenotype (OC behavior) and observed variables (Rumination, Contamination, and Checking) is constrained to be 1. The proportions, the square of the parameters of Figure 1, of genetic and environmental influences from the best fitting common pathway model are given in Table V.

For the latent OC behavior construct, 36% of its variance was attributed to genetic factors (A) and the rest of the variance was explained by nonshared environmental factors (E). Shared environmental factors (C) could be dropped without any loss of fit, which means that the influence of shared environmental factors is zero on the latent OC construct, and this factor is not shown in Figure 1. For clarity, a CE model also fitted the data ($\chi^2(1) = 1.4$, $P = 0.23$), though worse than the AE model. Dropping both A and C resulted in a significantly worse fit ($\chi^2(2) = 13.5$, $P = 0.001$). The latent OC behavior phenotype explained more than half of the variation of Rumination (56%) and Checking (69%), but interestingly only 25% of Contamination. So 75% of the variation of the Contamination dimension is explained by specific factors, with 33% explained by genetic factors and 42% by nonshared environmental

TABLE I. Results of Factor Analysis of 17 Padua Inventory Items Used in Present Study

	Factor			
	Rumination	Contamination	Impulses	Checking
Are you the type of person				
Who after doing something carefully, still has the impression it is either done badly or is not finished?	**1.000**[a]	0.083	−0.246	−0.111
Who has to do things several times before thinking they are done properly?	**0.687**	0.107	−0.101	0.203
Who imagines that catastrophic consequences may result from absent-mindedness or minor errors you have made?	**0.648**	−0.069	−0.013	0.150
Who invents doubts and problems about most of the things you do?	**0.562**	−0.148	0.231	0.089
Who has unpleasant thoughts that come into your mind against your will, and which you cannot get rid of?	**0.372**	−0.066	0.305	0.231
Who finds it difficult to touch garbage or dirty things?	0.083	**0.850**	0.087	−234
Who finds it difficult to touch an object which has been touched by strangers or certain people?	−0.007	**0.750**	−0.047	0.184
Who has to wash their hands more often and longer than necessary?	−0.030	**0.689**	0.038	0.173
Who avoids using public toilets because of fear of disease and contamination?	−0.025	**0.582**	−0.015	0.373
Who sometimes has to wash or clean yourself because you think you may be dirty or "contaminated"?	0.108	**0.474**	0.043	0.341
Who when looking down from a bridge or very high window, feel an impulse to throw yourself into space?	−0.240	−0.210	**0.964**	0.341
Who when a train approaches, sometimes thinks of throwing yourself under the wheel	−0.156	0.238	**0.940**	−0.259
Who, while driving, sometimes feels an impulse to drive the car into someone or something?	0.110	0.063	**0.795**	−0.104
Who sometimes feels a need to break or damage things for no reason?	0.308	0.061	**0.438**	−0.066
Who checks and rechecks gas burners, water faucets, and light switches after turning them off?	0.054	0.050	−0.003	**0.799**
Who has to return home to check doors, windows, and drawers etc., to make sure they are properly shut?	−0.027	0.182	0.009	**0.761**
Who has to keep on checking forms, documents, checks etc. in detail to make sure they have been filled out correctly?	0.220	0.046	−0.064	**0.695**

[a]The numbers represent the factor loadings on the four factors and bold numbers are the primary loadings on that factor.

factors. The shared environmental factor explained 0% of the variance and could be dropped without any worsening of the fit and is therefore not shown in Figure 1. For both the Rumination and Checking dimensions, genetic and shared environmental specific factors could be dropped without a significant decline in fit ($\chi^2(2) = 0.53, P = 0.77$, and $\chi^2(2) = 2.42$, $P = 0.30$, respectively), meaning that specific familial factors do not play a role in these two OC dimensions.

DISCUSSION

This is the first twin study to investigate genetic and environmental effects on different dimensions within OC symptoms in a population-based sample. We first completed a factor analysis on 17 PI-R items to search for distinguishable OC dimensions. We then completed multivariate twin analyses of three OC dimensions. Three main conclusions can be drawn. First, using the items of the PI-R of a population based sample of female twins in a factor analysis, four OC dimensions could be identified: Rumination, Contamination, Checking and Impulses. Second, using three of the four dimensions in the genetic analyses the common factor model best fitted the data, which means that there is a common OC behavior phenotype explaining variance of all three dimensions, and this phenotype is influenced by genes and nonshared environment. Third, besides genes for the broad OC behavior phenotype, specific genetic influences are also seen for Contamination dimension, explaining a fair amount of its variation.

The factor structure of the PI items we found was similar to that found in earlier studies using PI items within OCD patients [Van Oppen et al., 1995; Denys et al., 2004] and general population samples [Van Oppen et al., 1995; Burns et al., 1996]. We could not identify the dimension related to precision because no corresponding items were included in this study.

The results of this multivariate analyses show the extent to which symptom dimensions that cluster share a common

TABLE II. Pairwise Correlations (Within Person) Between OC Dimensions

	Rumination	Contamination	Checking
Rumination	1.00		
Contamination	0.30*	1.00	
Checking	0.57*	0.37*	1.00

*$P < 0.01$.

TABLE III. Twin Correlations Per OC Dimension

	Twin 1		
Factor	Rumination	Contamination	Checking
Twin 2			
Rumination	0.25/0.11	−0.21	0.20
Contamination	0.12	0.42/0.05	0.05
Checking	0.23	0.15	0.35/0.28

Correlations for MZ twins and DZ twins are reported below and above diagonal respectively. On diagonal, correlations for MZ twins are reported on the left and for DZ twins on the right.

TABLE IV. Model Fitting Results for Heritability of YASR-OC Dimensions

Number of model	Type of model	−2LL	χ^2	df	*P*	Parameters	Compared with model
1	Fully saturated model	4,683.0	—	—	—	42	—
2	Full independent pathway model	4,717.4	34.4	24	0.08	18	1
3	Full common pathway model	4,723.4	6.0	4	0.20	14	2
4	Common pathway model with common AE[a]	4,723.4	0	1	1.00	13	3
5	Common pathway model with common CE	4,724.8	1.4	1	0.23	13	3
6	Common pathway model with common E	4,738.3	13.5	2	0.001	12	3

A, additive genetic effects; C, common or shared environmental effects; E, nonshared or individual-specific effects.
[a]Best fitting model.

genetic basis. The common factor model fitted the data the best. The common factor, that is, OC behavior phenotype, was influenced by both genetic and nonshared environmental influences. Twin studies of OCD in adults so far also found no evidence for shared environment [Van Grootheest et al., 2005]. Our results further indicate that, in addition to a common factor, sharing genes related to three dimensions, only the contamination dimension may possess also specific genetic factors, while for the other two dimensions we have to conclude that specific familial influences are not of importance. Interestingly, the Contamination dimension is also the dimension of which only a quarter of the variation is explained by the common OC behavior phenotype. This means that the Contamination dimension is a relative independent dimension.

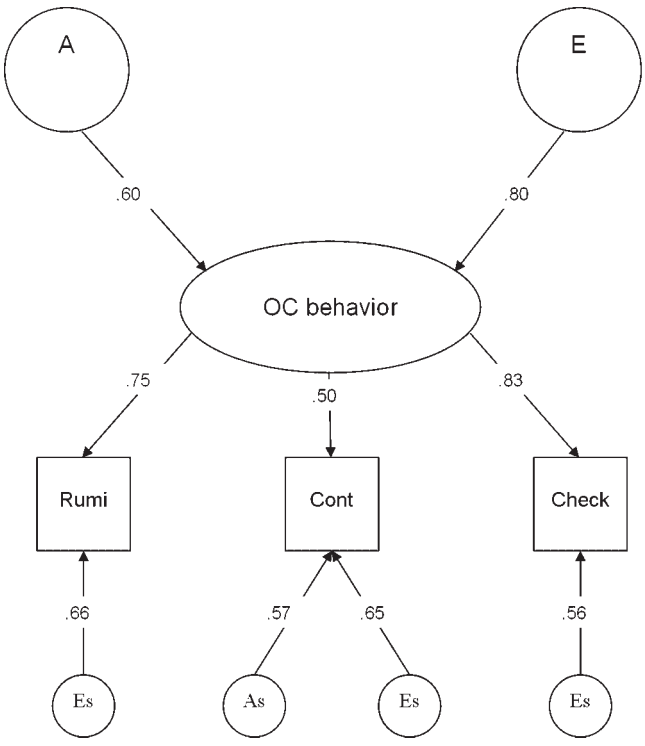

Fig. 1. Path diagram of the final common pathway model. Rumi, rumination; Cont, contamination; Check, checking; A, additive genetic; C, shared environment; E, nonshared environment; As, specific additive genetic; Cs, specific shared environment; Es, specific nonshared environment; OC behavior, latent phenotype. The numbers reflect path coefficients. The square of the path coefficients is the proportion of explained variance. The total variance of the latent phenotype (OC behavior) and observed variables (rumination, contamination, and checking) is constrained to be 1 (i.e., four constraints). For example, variance of Rumination is 0.66×0.66 $(=0.44) + 0.75 \times 0.75$ $(=0.56) = 1$.

These results support the findings of some of the family studies investigating the familiality of OC symptom dimensions, based on Y-BOCS items [Leckman et al., 2003; Hasler et al., 2007]. Hasler et al. [2007] found significant sib–sib correlations for Checking compulsions and the Contamination/Cleaning dimension. These two Y-BOCS dimensions are comparable with the PI-R Checking and Contamination dimensions we found. However, we found no familial effects for the Checking dimension, but the study of Hasler et al. [2007] did not account for the possibility of common versus specific familial effects.

These results suggest that, in spite of clinical heterogeneity, a broad OC behavior phenotype exists, influenced by both genes and nonshared environment. This corresponds well with clinical presentations of OCD: OCD patients typically score positive on a wide variety of symptoms from multiple dimensions, with usually one or two dimensions appearing more prominent [Leckman et al., 1997]. Our results seem also in line with Mathews et al. [2004] who examined the structure of OC symptoms in a nonclinical population and concluded that this broad OC behavior phenotype, they call it "obsessionality," is phenomenologically similar to OCD and is likely to comprise a continuum with OCD. This may implicate that, besides a traditional categorical model of OCD, an underlying quantitative OC behavior phenotype could be used to provide an alternative strategy for the detection of genetic susceptibility loci that contribute to OCS or OCD [Miguel et al., 2005]. Another approach would be the use of the contamination dimension, showing clear specific genetic influences explaining a substantial amount of its variance.

These results should be interpreted in the context of four limitations. First, we only selected a subset of items from the PI which probably increased total error variance. Error variance cannot be distinguished from nonshared or individual-specific environment, and therefore it is likely that the impact of genetic influence on the etiology of OCS is underestimated. Second, the present study only included women, so results cannot be assumed to hold equally for males, although Van Grootheest et al. [2007] recently found in a large twin-family study, that the same genetic risk factors were expressed in men and women for OC behavior. Third, because of the use of a threshold model [Derks et al., 2004], and the fact that number of MZ twins exceeded the number of DZ twins [Posthuma and Boomsma, 2000], the power to distinguish genetic influences from shared environmental influences was moderate. Fourth, the findings of this analysis are predicated on the assumptions of the method used. These assumptions include no large degree of assortative mating and the validity of the equal environment assumption (EEA). The EEA states that environmental influences are shared to the same extent by MZ and DZ twins. Maes et al. [1998] found that significant but moderate primary assortment exists for psychiatric disorders. However, it was concluded that the bias in twin studies caused by the small amount of assortment is negligible. Jonnal et al. [2000] tested

TABLE V. Proportions of Variance Explained by Genes and Environment From Best Fitting Common Pathway Model

Latent phenotype			Common pathways[a]		Specific pathways[b]		
	A	E			A	C	E
OC behavior	0.36	0.64	Rumination	0.56	—	—	0.44
			Contamination	0.25	0.33	—	0.42
			Checking	0.69	—	—	0.31

A, genetic influences; C, shared environmental influences; E, nonshared environmental influences.
[a]Variation shared by the latent phenotype (OC behavior) and the specific factors (OC dimensions). Proportions of common pathway and specific pathways add up to 1 for each factor.
[b]OC factor or dimension specific contributions.

the EEA for OC symptoms in the current sample and concluded that the EEA was not violated.

The limitations of the present study give direction for future twin studies investigating OC dimensions. First step is to replicate our results in a large twin sample with an adequate MZ/DZ twin ratio to overcome power limitations [Posthuma and Boomsma, 2000]. Second, assessing OC symptoms in both male and female twins allows one to test for sex-differences within symptom dimensions. Finally, it is preferable to assess OC symptoms with the complete PI-R and/or Y-BOCS [Goodman et al., 1989]. The relatively new self-report version of the Dimensional Y-BOCS (DY-BOCS) [Rosario-Campos et al., 2006], especially developed to assess OC dimensions, seems promising in this respect. Using both the (D)Y-BOCS and PI-R has the advantage of assessing unique factors: Rumination is represented solely in the PI-R and "somatic/religious/sexual obsessions" and "hoarding obsessions/compulsions" solely in the (D)Y-BOCS.

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